Behavioral impairment produced by low-level postnatal PCB exposure in monkeys.

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The preponderance of evidence in humans suggests that polychlorinated biphenyl (PCB)-induced behavioral deficits result from prenatal exposure rather than exposure through breast milk, although a recent study reported lower psychomotor scores during infancy associated with PCB concentration in breast milk. In the current study, monkeys were dosed from birth to 20 weeks of age with a PCB congener mixture representative of the PCBs found in human breast milk. Blood and fat levels of PCB-exposed monkeys at the end of the dosing period were within the range observed in the general human population, while levels in control monkeys were below averages observed in humans in industrialized countries. Behavioral assessment on a series of tasks was performed when monkeys were between 2.5 and 5.0 years of age. Robust deficits were observed on spatial delayed alternation, fixed interval, and differential reinforcement of low rate performance. No group differences were observed for the number of errors on a series of nonspatial and spatial discrimination reversal tasks. Behavioral deficits included retarded learning, perseverative behavior, and inability to inhibit inappropriate responding. These results have implications for the potential contribution of exposure to PCBs through breast milk to behavioral impairment. Copyright 1999 Academic Press.

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